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EXOTIC NEWCASTLE DISEASE ACTIVITIES



On July 25, 1977, exotic Newcastle disease was confirmed in a retail pet shop in Kaneohe, Oahu, Hawaii, when the virus was isolated and characterized from specimens submitted from two Amazon parrots. The specimens were submitted by the Hawaii Department of Agriculture Veterinary Laboratory, Honolulu, Hawaii, to Veterinary Services Laboratories (VSL), Ames, Iowa, where the virus was isolated.

The two Amazon parrots from which the virus was isolated, were part of a shipment of 16 parrots which were shipped on June 21, 1977, from a commercial aviary in Bakersfield, California. Four parrots had previously been shipped on June 14, 1977. The second shipment of birds from the premises in Bakersfield, California, to the retail shop in Hawaii included a total of 16 Amazon parrots. Fifteen of these birds died. The remaining bird was purchased and destroyed. Several of the birds in the June 21, 1977, shipment

were sampled and specimens were submitted to VSL, Ames, Iowa. The virus of exotic Newcastle disease was isolated from several of the birds.

The premises in Hawaii which received the infected birds and the premises in Bakersfield, California, were immediately placed under State "hold" orders. On July 29, 1977, the retail shop and one other aviary in Hawaii were placed under Federal quarantine. The birds in the pet shop in Hawaii were appraised and humanely depopulated and the premises were cleaned and disinfected. Birds from the pet shop in Hawaii which were sold since June 15, 1977, the date that the first shipment of birds arrived from the Bakersfield bird dealer, were traced and an epidemiological evaluation was conducted.

On July 28, 1977, 136 birds in the commercial aviary at Bakersfield were swabbed and these swabs as well as four dead birds were submitted to the San Gabriel, California State Diagnostic Laboratory. On August 7, 1977, the virus of exotic Newcastle disease was isolated and characterized from five of the specimens submitted. The birds in the aviary at Bakersfield were appraised, humanely destroyed, and the establishment cleaned and disinfected and vacated for 30 days. A Federal quarantine was placed on the premises in Bakersfield on August 9, 1977. Birds sold from the shop in Bakersfield since June 4, 1977, were traced and an epidemiological evaluation conducted.

The source of the birds which were purchased by the dealer in California on June 4, 1977, was not definitely determined. Some evidence indicates that these birds may have entered the United States illegally. Continued surveillance indicates that there was no spread of the disease to poultry or to other aviaries.

On September 7, 1977, the quarantines were lifted in Hawaii after surveillance and evaluation had been made. The quarantine on the premises in California was lifted on September 15, 1977.

TWENTY-SECOND SESSION OF THE EUROPEAN COMMISSION FOR THE CONTROL OF FOOT-AND-MOUTH DISEASE

The Twenty-Second Session of the European Commission for the Control of Foot-and-Mouth Disease (ECCFMD) was held in Rome, Italy, from March 29-April 1, 1977. Delegates of some 22 member countries attended, along with observers from international organizations and other countries. USDA veterinarians from both the Animal and Plant Health Inspection Service and the Agricultural Research Service were among the observers.

The ECCFMD has both an executive committee and a research group which meet frequently between the biennial open sessions in Rome. The Secretariat is the executive agency given the Commission's operational responsibility in Europe and other areas of interest to the Continent. As seen necessary, emergency sessions, called "ad hoc consultations" are convened. Reports of activities of the Secretariat, the Executive Committee, and the Research group were presented.

There was a decline in disease during the last 2 years, as there has been during the history of the ECCFMD. Even with this decline there was no complacency during the discussions of the position and control of FMD in Europe. Italy, a large live-animal and meat importer from many countries, continued to record significant outbreaks of disease.

A number of recent FMD cases throughout Europe have been due to accidents, either laboratory escape of virulent virus or use of incompletely inactivated vaccines. Because of virtually complete annual vaccination of cattle with trivalent OAC type vaccines in continental western Europe, spread of disease is usually limited.

Europe's proximity to Asia and Africa requires constant vigilance and effort. Through both the Food and Agriculture Organization (FAO) and the European Economic Commission (EEC), Europe supports FMD prophylaxis in southeastern Europe, particularly against exotic types and subtypes, such as Asia₁ and A₂₂, against which routine vaccination would offer no protection should these viruses enter. Work is coordinated by a tripartite FAO/EEC/OIE (Office of International Epizootics) Committee which meets periodically.

Greece, Bulgaria, and the European part of Turkey vaccinate the bulk of Thrace (southeastern Europe) against these exotic viruses. These areas, which are the buffer zone for the European Continent, have recorded no exotic disease during the previous 2 years.

A new development in northeastern Africa was of immediate concern to the delegation. FMD in nearby Morocco, after absence of the disease for more than 10 years,

had become epidemic and had spilled over into neighboring Algeria. Though European vaccines had been used, the Commission had still received insufficient information about whether or not their conventional European vaccines would hold against this new A subtype.

The Commission reviewed a Pan American Health Organization (PAHO) Rio de Janeiro center report of FMD in South America, from which Europe imports beef. Concern was expressed about insufficient knowledge of the classification of new strains present in South America. APHIS described its cooperative FMD prevention efforts with Central America and Colombia.

The Swine Vesicular Disease (SVD) situation in Europe was discussed with Great Britain reviewing its ambitious eradication program which cost \$15 million. A Plum Island Animal Disease Center (PIADC) scientist told of work showing the SVD virus' very close relationship to the coxsackie B5 virus infecting humans. He also described its extreme hardiness in pork products, and a serological survey showing no evidence of SVD presence in the United States.

Some other subjects discussed at the meeting were the strengthening of FMD laboratory infrastructure, the holding of emergency stocks of exotic seed virus, the significance of the "carrier state" of FMD, a joint study for the evaluation of methods of virus and antibody assays carried out by 18 world laboratories, including PIADC, and the important evolution toward standardization of measuring vaccine potency.

PACHECO'S PARROT DISEASE

Following the 1929-1930 psittacosis pandemic in Europe and the United States, a team of investigators, Pacheco and Bier, observed psittacosis in Brazil, particularly in Brazilian parrots.¹ In addition to the isolation of the agent of psittacosis, they found a filterable virus which seemed to be involved in at least one of the morbid conditions. The virus was transmitted to Tui parakeets without difficulty, while attempts to transmit the infection to guinea pigs, mice, chickens, pigeons, and monkeys were unsuccessful. In 1931, laboratory studies were conducted on the Brazilian isolate by Rivers and Schwenker at the Rockefeller Institute. This study revealed that the cause of the parrot mortality was a virus unrelated to psittacosis and from microscopic observation it was tentatively identified as a herpes virus. In 1933, Findlay designated this condition as Pacheco's parrot disease. In 1975, the disease was diagnosed in Kenya, as well as in psittacine birds in Florida.²

Identification of the agent as a herpes virus was based on its characterization by electron microscopy, sensitivity to ether, filtration studies, and the presence of intranuclear inclusion bodies in liver cells of experimentally infected chick embryos. In transmission studies, budgerigars were used very successfully. The clinical disease was manifested by ruffling of feathers, and progressive weakness, terminating in death within 3-7 days. The nasal discharge, diarrhea, and loss of weight, which are usually seen in psittacosis, were not particularly evident.

On post mortem examination, changes in the liver and spleen were the predominant features. The gross changes in the chick embryos were greenish discoloration of the livers, and either small focal or diffuse areas of necrosis.³

On histological examination, the affected livers from dead embryos and dead budgerigars revealed caseation necrosis of varied size. Multinucleated cells contained eosinophilic intranuclear inclusions in tissue sections stained with hematoxylin-eosin. Also, chromatin was markedly margined. Lipids were found accumulated at the periphery of necrotic areas. In addition, there was bile stasis in the livers of chick embryos. A similar pathological picture appeared in the spleen. Electron microscopic examination showed ultrastructural alterations of hepatocytes, intranuclear inclusions, lipid-like vacuoles in inclusions, margination of chromatin, karyorrhexis, as well as scattered and enveloped nucleocapsids in the nucleus and cytoplasm.

Unsuccessful attempts to protect budgerigars by vaccinating four birds with a lyophilized turkey herpes virus which is commonly used to protect poultry from Marek's disease, were conducted. Based on that very limited study, turkey herpes virus may not be effective as a vaccine for Pacheco's disease of psittacines.

In the differential diagnosis, possible confusion of Pacheco's disease with psittacosis and viscerotropic velogenic Newcastle disease (VVND) should be borne in mind. Pacheco's disease is fairly species-specific, affecting psittacine birds. In general, however, it seems that nasal discharge, anorexia, diarrhea, and loss of weight are less pronounced in this disease than in psittacosis, probably due to the acuteness of Pacheco's disease. In addition, psittacotic infection produces minute basophilic coccoid bodies in the cytoplasm.

The intracerebral inoculation of Pacheco's virus appeared to induce a septicemic form of the disease without definite brain lesions. Several passages through parakeets failed to intensify the pathogenicity of the virus. Only 1 or 2 day old chickens manifested meningitis following injection of virus, although serial passages in young chickens were not attempted. This observation indicates that young chickens are at least slightly susceptible to the Brazilian virus, while chicken embryos inoculated with this agent died usually on the fifth day after inoculation. Acute bird losses, which were attributed to Pacheco's disease, have been reported. It was commonly indicated that the birds were fairly normal in the morning, found on the floor of the cage in the afternoon, and dead that evening. There is no known cure or treatment for the condition.

In contrast to the herpes virus of Pacheco's disease, VVND can produce high mortality in young susceptible birds, a very short course of disease (5-9 days), signs of diarrhea, swelling of head and neck, and hemorrhagic lesions in the digestive tract. The agent of VVND is lethal for chicken embryos, and produces hemagglutinins in the allantoic fluids that can be inhibited by Newcastle disease immune sera. The Pacheco's disease agent is non-hemagglutinating.

Work is being conducted on the isolation of Pacheco's disease at Veterinary Services Laboratories, Ames, Iowa. At least 17 lots of birds submitted to the laboratory have yielded the non-hemagglutinating, chick embryo lethal, chick kidney cell culture cytopathogenic agent which has been identified by an electronic microscope as a herpes virus.

The clinical picture of VVND in pet bird species is different from that observed in Pacheco's disease. In observing the course of VVND in six pet bird species,

Erickson noted that the clinical response to a psittacine isolate of VVND was most marked in budgerigars, parrots, and conures. A minimal response was noted in black-headed nuns. By 3-5 days following exposure, some birds had ruffled plumage, conjunctivitis, and evidence of central nervous system damage. The symptoms of the nervous system were ataxia, wing tremors, paralysis, tremors of head, nodding, and jerking movements. Visceral lesions in the posterior gut, commonly seen in chicks, were not observed in budgerigars, parrots, canaries, conures, mynahs, and nuns.⁴

¹Pacheco, G., and Bier, O.:Epizootia Em Papagaios No Brasil E Suas Relacoes Com A Psittacose. Arch. Inst. Biol. Sao Paulo, 4:89, 1931.

²Simpson, Charles F., and Hanley, J. E.:Pacheco's Parrot Disease in Psittacine Birds. Avian Diseases 21(2):209-219, 1977.

³Rivers, Thomas M., and Schwentker, Francis F.:A Virus Disease of Parrots and Parakeets Differing from Psittacosis. J. Exp. Med., 55:911-924, 1932.

⁴Erickson, G. A.:Viscerotropic Velogenic Newcastle Disease in Six Pet Bird Species:Clinical Response and Virus-Host Interactions (Ph.D dissertation).

CONTAGIOUS EQUINE METRITIS

Reports on a condition termed "equine vaginitis" were received by the U.S. Department of Agriculture (USDA) from the United Kingdom (U.K.) and Ireland in late May and June of 1977.

On August 12, 1977, Australia placed a ban on horses from the U.K. and Ireland, to terminate November 1, 1977. On September 1, 1977, Veterinary Services of USDA's Animal and Plant Health Inspection Service, Hyattsville, Maryland, the Plum Island Animal Disease Center (PIADC), and the American Association of Equine Practitioners sent three veterinarians to England and Ireland. In addition, Veterinary Services European/African representative visited France.

The following summarizes the information obtained in the U.K. and Ireland: The present name of this disease is contagious equine metritis (CEM). It is a venereal disease caused by a bacteria which produces serious economic losses in thoroughbred breeding establishments. To date, CEM has only been reported in thoroughbreds.

An outbreak of CEM occurred in Ireland during the breeding season of 1976 in 10 stud farms. It spread to the Newmarket area of England and appeared during the breeding season of 1977. It was reported to have affected three stud farms in Ireland (in 1977) and 17 among 40 stud farms in Newmarket, England. The Newmarket losses were estimated to be approximately \$30 million. Among the mares bred in the affected stud farms in Newmarket, 30 percent were said to have shown clinical signs of the disease.

The primary clinical manifestations in mares are: 1) A copious, purulent vaginal discharge occurring 3-5 days after mares have been bred. Many mares return to estrus due to lack of conception. Some mares experience early abortions. It is believed a few infected mares can carry foals to term; 2) Transmission can occur when infected stallions breed mares or contaminated instruments are used to examine the genital tract of mares, or by other means such as washing the genital

area of the infected mares using the same water and sponge among non-infected mares.

On September 9, 1977, the USDA placed a temporary ban on the importation of all horses and other equidae from Ireland, U.K., and France. On September 16, 1977, a similar ban was placed on Australia since CEM had just been reported at a stud farm in the State of Victoria. On September 22, 1977, weanlings and geldings were declared to be exempt from the ban by the USDA.

Full information on the extent of CEM in France has not yet been received.

It is evident that horses have entered the United States from affected countries prior to September 9, 1977. These imported animals will be investigated and pertinent history will be obtained to evaluate their potential danger. It is planned that appropriate animals will be cultured to aid in determining their disease status.

It is of utmost importance that United States horse owners and veterinarians carry out diligent steps to diagnose any cases of CEM and come to grips with the problem to prevent its spread among our breeding stock before it is firmly established.

The causative agent of CEM appears to be a microaerophilic gram-negative coccobacillus that is similar to, but not identical with, moraxella, brucella, and hemophilus organisms. The Collindale Typing Center in England made the foregoing determination.

The microaerophilic nature of this bacteria caused delay in successful culturing of this causative agent in England. Increased requirement for carbon dioxide and reduced oxygen tension are important aspects of culturing this new bacteria. Diagnostic assistance is available at the USDA, PIADC.

When suspicious mares or stallions are encountered, assistance with culturing can be obtained by contacting State or Federal Animal Health officials.

Experimentally, transmission of CEM seems to be enhanced in the presence of stallion semen. Limited work relating to necropsies of stallions has not shown gross pathological changes.

Chemotherapy against the causative agent of CEM has involved numerous drugs, but ampicillin and other penicillin derivatives seem to be most practical. Controlled work directed at defining chemotherapeutic success needs to be carried out.

Research on CEM will be carried out at USDA, PIADC to complement research that has been completed and is presently underway in England and Ireland.

It should be recognized that CEM is not "vicious" in its attack on an individual animal, that is, the lesions are superficial and systemic infection is not evident. The important feature of CEM is that it is highly contagious. This contagiousness has a devastating effect on the reproductive efficiency in horses when coupled with the seasonal breeding limits existing in mares.

WORLD DISEASE REPORTS*

Country	Date 1977	New Outbreaks	Country	Date 1977	New Outbreaks
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Foot-and-Mouth Disease

Argentina	February 1-15	4	Jordan	June-Sept. 1976	5
Bolivia	January-March	71	Kenya	February-June	10
Botswana	February	5	Kuwait	March-May	27**
Brazil	January-June 17	4,994	Morocco	March-June	168
Burundi	January-April	7	Nigeria	Oct.-Dec. 1976	5
Cameroon	January-April	5		April-May	2
Chad	December 1976	1	Rhodesia	March	1
Chile	April-June	11	Rwanda	August 1976	1
Colombia	January-February	36		Oct.-Nov. 1976	4
	March	11**	Senegal	Jan.-Dec. 1976	10
	April-June	27	South Africa	June	2
Ecuador	February	1	Spain	January-April	19
Egypt	March-May	2	Sudan	Aug.-Oct. 1976	9
Hong Kong	February-July	40	Syria	February	2
India	Sept.-Dec. 1976	973	Tanzania	December 1976	4
	January-March	1,002	Thailand	November 1976 -	
Iran	February-June	21		March 1977	63**
Iraq	February-March	13	Turkey	February-June	389
	May-July	7	Upper Volta	Oct.-Dec. 1976	1
Italy	May-June	3	U.S.S.R.	January-May	55

Sheep Pox

Chad	April	1	Kuwait	March-May	48**
Egypt	June 16-July 15	4	Libya	March-April	23
India	Sept.-Oct. 1976	7	Morocco	March 16-June 30	15
Iran	February-June	39	Portugal	January-February	1
Iraq	February	94	Senegal	Jan.-Dec. 1976	1
	March	31**	Sudan	July-Oct. 1976	25
	May-July	32**	Syria	February	54**
Israel	February	1		April	21
	April	1	Tunisia	March-May	2
Israel (control territory)			Turkey	January-June	445
	February-March	10	U.S.S.R.	January-February	3
	May	1			

Contagious Bovine Pleuropneumonia

Cameroon	April	1	Kuwait	March-May	15**
Chad	December 1976 -		Niger	January-April	11
	May 1977	9	Nigeria	April-June	7
Ghana	January-April	18	Senegal	Jan.-Dec. 1976	1
Ivory	December 1976 -		Sudan	July-Oct. 1976	28
Coast	January 1977	4	Upper Volta	October 1976 -	
				March 1977	6

Country	Date 1977	New Outbreaks	Country	Date 1977	New Outbreaks
<u>Lumpy Skin Disease</u>					
Botswana	February-April	20	South		
Congo(Pop. Rep.)	July	1	Africa	February-June	107
Madagascar	Aug.-Dec. 1976	11	Swaziland	January-June	3
	January-April	31			
<u>African Horse Sickness</u>					
Senegal	Jan.-Dec. 1976	1	South		
Sudan	July-Oct. 1976	4	Africa	March-June	15
<u>African Swine Fever</u>					
Congo(Pop. Rep.)	July	1	South		
Portugal	March-July	304	Africa	February-June	3
Spain	Feb. 16-June 15	384	Zaire	January-March	1
<u>Rinderpest</u>					
India	Sept.-Dec. 1976	31	Sudan	July-Oct. 1976	8
	January-March	40			
<u>Dourine</u>					
South Africa	February-June	11			
<u>Teschen Disease</u>					
Austria	March-May	4	Madagascar	Aug.-Dec. 1976	15
				January-April	19
<u>Swine Vesicular Disease</u>					
Italy	March-August 15	9	United Kingdom	March-June	4

(*Extracted from International Office of Epizootics, Monthly Circular, numbers 364, 365, 366, 367, and 368).

(**Cases).